Trauma to the Urinary Tract

SIR,—Mr. J. P. Mitchell's paper (5 June, p. 567) succeeds admirably in its declared object of seeking to clarify and simplify this variegated subject. The more conservative approach to kidney trauma has truly emerged in the last two decades; nephrectomy is rarely the method of choice in its treatment. Mr. Mitchell rightly postulates a quandary for the surgeon—the decision confronting him whether or not to operate, and when—if he does so.

In addition to the more usual, there is one little-recognized type of renal injury which provokes particularly anxious deliberation both in diagnosis and in treatment. This is when a direct blow to the region causes extensive damage to the parenchyma but is unaccompanied by any cortical tear; in other words formation of a subcapsular haematoma. With no tension-avoidance allowed by any superficial rent there is clinically persisting pain and tenderness in the renal angle. There may be no haematuria. When some 48 hours have elapsed intravenous pyelography will show distortion of the caliceal pattern. It is possible that conservation may succeed and the haematoma be absorbed. But if no relief to the intrarenal tension is forthcoming then complete destruction of the organ is also feasible. This comes about by a process of autolysis which can take a span of six months; the kidney may be transformed into an inert bag of pultaceous, macerated parenchyma admixed with blood elements. Infection can readily supervene under these circumstances. The decision whether such a closed injury should be decompressed by incision in the capsule is one calling for a high degree of experience and judgement. On two occasions I have had the unhappy experience of removing such functionless remnants and because of that I could be biased towards intervention if I felt reasonably assured of the precise diagnosis.

I also wonder if these two defunct kidneys had been left alone would they have become, after an intermediate hydrocalycosis stage, the shrunken pathological specimens like the one itemized in Mr. Mitchell's follow-up of 56 cases? Was his the final remnant of an autolytic process?—I am, etc.,

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Radiologists in Liverpool

SIR,—Following the "Contemporary Themes" article by Professor A. G. W. Whitfield and others on "Radiological Training and Staffing in the Birmingham Region and Elsewhere" (29 May, p. 514), I have now analysed the consultant radiological appointments made in the Liverpool Regional Board hospitals during the ten-year period January 1961 to December 1970.

Twelve consultant appointments were filled, eleven by locally trained radiologists. Five of the appointees had obtained the Fellowship of the Faculty of Radiologists, with two further consultants obtaining this diploma shortly after appointment, This figure (58%) with the F.F.R. is greater than that of the Birmingham R.H.B. hospitals and is also considerably higher than that of consultants appointed in Liverpool before the survey period.

At first sight it would appear that the Faculty of Radiologists is being successful in raising the standard of training for consultant posts throughout the country, but it is of interest to carry the analysis one step further. All five consultants appointed with the D.M.R.D. as their only radiological diploma are still working in Liverpool R.H.B. hospitals, though one consultant has changed his appointment. Only three of the consultants appointed with the F.F.R. have remained in Liverpool R.H.B. hospitals. Of the four consultants who have left, two have subsequently been appointed to teaching hospitals in Liverpool and the other two have emigrated to Canada and are now working in Canadian teaching hospitals.

How different is the situation in radiology compared with other branches of the profession. One rarely hears of consultant physicians or surgeons in R.H.B. hospitals changing their appointments, or emigrating overseas to greater financial opportunities. Emigration in these branches usually occurs at senior registrar or registrar levels.

It would be of interest to know if this is the pattern in radiology throughout the country and why such a high proportion of consultants with the F.F.R. leave the regional hospital board posts.—I am, etc.,

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Treatment of Myasthenia

SIR,—In your "Today's Drugs" article on the treatment of myasthenia gravis (17 April, p. 160) your author states that thymectomy carries a relatively high morbidity and mortality.

I think your author's statistics are out of date. Since the practice was begun several years ago of discontinuing anticholinesterase medication during and in the immediate post-thymectomy period, as well as the routine use of tracheostomy, the mortality in most centres has been quite small.[1]—I am, etc.,

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1 Papatestas, A. E., Alpert, L. I., Osserman, K. E., Osserman, R. S., and Kark, A. E., *American Journal of Medicine*, 1971, **50**, 465.

Aortic Aneurysm and Peptic Ulcer

SIR,—In connexion with your leading article on "Aortic Aneurysm and Peptic Ulcer" (16 January, p. 129) it may be of interest that our observations made recently (to be published partly in *Polish Medical Weekly*) show an association between peptic ulcer and generalized arteriosclerosis with renal artery stenosis and hypertension in males in their thirties and forties. We have observed seven males aged 30-48 years with renal artery stenosis, hypertension, and signs of general arteriosclerosis (claudication, diffuse neurological lesions, etc.), in five of whom there was a history of peptic ulcer.

This remark broadens, I hope, the observations made by A. Elkeles[1] about the association between radiological calcification of the aorta and its branches and gastric ulcer in people over the age of 50.—I am, etc.,

W. WERMUT

Medical Academy, Gdańsk, Poland

1 Elkeles, A., *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine*, 1964, **91**, 744.

Malaria and Sickle-cell Disease

SIR,—The interesting article of Mr. A. Adeloye and colleagues (22 May, p. 445) corrects a longstanding impression that *all* sicklers (whether they have the trait or the disease) are protected against severe malaria.

In a 5-year study of 1,391 consecutive sickle-cell disease patients (Table 1) sickle-cell crisis (as defined by experienced workers in this field[1 2]) severe enough to require admission to hospital occurred in 281 (42·7%) of the 658 Hb SS patients, 211 (31·8%) of the 664 Hb SC patients, 14 (40%) of the 35 Hb S β thalassaemia, 8 (25%) of the 32 Hb $SF_{highgene}$, and none of the 2 Hb SF_{Punjab} disease patients. Precipitating causes of crisis severe enough to require emergency admission to Korle-Bu Hospital, Accra, included malaria, upper respiratory tract infection, pneumonia, enterocolitis, salmonellosis, urinary tract infection, pregnancy, and miscellaneous ones like trauma, severe exercise, alcoholic intoxication, and heavy rainfall. In 75 (26·6%) of the 281 Hb SS patients admitted in crisis the cause was diligently looked for but not found. The corresponding number of Hb SC patients admitted in whom the cause of crisis was unknown is 62 (29·4%) out of 211. The number of patients with malaria infection (usually mixed, with *P. falciparum* predominating) causing crisis severe enough for admission to hospital is given in Table II for the two largest genotypes. While malaria severe enough to cause serious crisis is common in sickle-cell disease (8·2% of 1,322 patients) it is three times the cause of crisis admissions in sickle-cell anaemia as in Hb SC disease (Table II, column percentages), and whereas 82 (12·5%) of all Hb SS patients were admitted in crisis because of

TABLE I.—*Sickle-Cell Disease Patients Studied*

Genotype	Number
SS	658
SC	664
S β thalassaemia	35
$SF_{highgene}$	32
SD_{Punjab}	2
Total	1,391

TABLE II.—*Sickle-Cell Disease Patients* Admitted in Crisis with Malaria*

Genotype	Crisis with malaria admission		Total
	Yes	No	
SS	82 (12·5%) (75·9%)	576 (87·5%)	658 (100%)
SC	26 (3·9%) (24·1%)	638 (96·1%)	664 (100%)
Total	108 (8·2%)	1,214 (91·8%)	1,322 (100%)

Chi-Square : 31·1 Degrees of freedom : 1

*10 of the 82 Hb SS patients were admitted twice with malaria causing crisis, 2 three times, and 1 four times. Only one of the 26 Hb SC patients was admitted twice with malaria precipitating crisis.

malaria only 26 (3·9%) of about the same number of Hb SC patients were admitted under similar circumstances. The results are very highly significant ($p<0.001$).

Though parasite counts are said to be lowest in sickle-cell homozygotes, intermediate in heterozygotes, and highest in normal homozygotes[3,4] suggesting, perhaps, that malaria in sickle-cell anaemia patients is not severe, it is my view (and that of Edington[5]) that in Ghana malaria in sickle-cell anaemia patients can be very severe, and is a serious common precipitating cause of crisis. I have also seen death result more than once from this. Apart from the mechanisms given by Mr. Adeloye and colleagues "whereby malaria might actually lead to death in the homozygote" the role of pyrexia per se in causing in vivo sickling is known, while the accompanying hyperhidrosis, anorexia, vomiting, and diarrhoea in young children can lead to serious dehydration with massive intravascular sickling, severe erythrocyte sequestration, and instant death.

G-6-PD deficiency has not been found to be statistically significant in the incidence of crisis admissions due to malaria in Accra. Thompson[6] found that while sickle-cell trait children were protected against *P. falciparum* sickle-cell trait adults were most often sick from this infection compared with Hb AC adults (lowest incidence) and normal homozygotes ($p<0.05$).

Without chloroquine many of the Accra sickle-cell disease patients would have died in crisis before now. There is no difference, mortalitywise, between the sickle-cell anaemia patient who is killed by malaria and the one who dies from crisis resulting directly from malaria. No sickler gets a crisis unless there is a change in the *milieu intérieur*, and there is usually a precipitating cause to produce this change. The maxim in Accra, as in Ibadan, is never merely to "treat a sickle-cell crisis" but to search quickly and diligently for the cause and then treat that.—I am, etc.,

I am grateful to Mr. R. G. Carpenter and Miss J. Nixon of the Department of Human Ecology, Cambridge, for their help with statistical analysis of data.

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1 Diggs, L. W., *American Journal of Clinical Pathology*, 1965, **44**, 1.
2 Edington, G. M., and Gilles, H. M., *Pathology in the Tropics*, p. 379, London. Arnold, 1969.
3 Vandepitte, J., and Delaisse, J., *Annales de la Société Belge de Medicine Tropicale*, 1957, **37**, 703.
4 Raper, A. B., *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 1959, **53**, 110.
5 Edington, G. M., *British Medical Journal*, 1953, 2. 957.
6 Thompson, G. R., *British Medical Journal*, 1962, 1, 682.

SIR,—I was very interested in the report from Ibadan of "Severe Malarial Infection in a Patient with Sickle-cell Anaemia" (22 May, p. 445). On several occasions while working at Awgu in the East Central State of Nigeria I had patients with sickle-cell anaemia who died during an attack of malaria. In at least two cases the diagnosis of the haemoglobinopathy had been confirmed by electrophoresis (at Enugu General Hospital).

The usual pattern was that after diagnosis children with the SS genotype were seen in outpatients instead of at baby welfare; they were kept on routine antimalarials (Daraprim) and folic acid, and the mother was encouraged to bring the child if he had any illness at all. In the fatal cases there was usually a period of regular attendance and then the family defaulted, to be seen next with a severely anaemic child (haemoglobin usually between 10% and 15%) with high fever. Malaria parasites were found in the blood.

Unfortunately I have to write from memory as our records were lost during the civil war, but I feel it is probable that an attack of malaria is often associated with the final illness of children with sickle-cell anaemia, and that as suggested in the article it is likely that the increased resistance to malaria is confined to the AS heterozygote.—I am, etc.,

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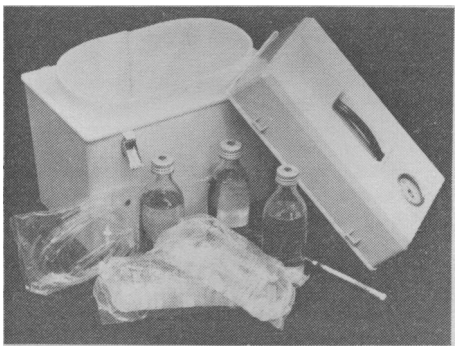
Insulating Kidney Perfusion Fluid

SIR,—As forecast by Calne[1] an increasing number of donor kidneys for transplantation are being supplied from hospitals peripheral to the transplantation unit. It is the responsibility of the transplant team to provide the staff and materials required to obtain these kidneys. These materials consist of pre-sterilized gowns, gloves, drapes, and instruments and a supply of cooled sterile perfusion fluids. All these materials must be unobtrusive yet readily available for time is a vital factor in obtaining donor kidneys.

Difficulties may arise in maintaining the perfusion fluids at a sufficiently low temperature and for this purpose we have used a modification of a commercially produced "cold box" (Nilo), size 16 in x 9 in x 14 in



(40 cm x 22·5 cm x 35 cm), which will normally maintain the temperature of its contents between 1° and 4°C for three to four hours. An extra layer of insulation in the form of a tailored inner lining of Evazote foam (6 mm thick) was added so that the contents of the box remained at the same low temperature for a longer period of time. To this insulated box is added three bottles of perfusion fluid, a giving set, and a low-reading thermometer and the whole unit stored unsealed in a cold room (4°C) in the transplantation unit.

When a donor kidney situation arises two 1 l. plastic packs of deep frozen saline (−15 to −20°C) (Allen and Hanburys Steriflex) are brought to the cold room and placed inside the box, which is then sealed. These not only provide a means of maintaining the low temperature within the box for at least 24 hours but also provide a supply of sterile frozen saline to charge the Thermos flasks used to transport the donor kidneys. In a series of experiments using thermocouples the temperature of the perfusion fluids remained between 1° and 4°C for a period of 24 hours. In fact the temperature of these fluids was still below 4°C after 36 hours but the amount of frozen saline remaining in the 1 l. packs was insufficient for use in filling the Thermos flasks.

However, for practical purposes a period of 24 hours is quite adequate for a donor kidney situation. By replacing the cold box and its contents daily a continuous supply of cold perfusion fluids and sterile iced saline can be maintained in close proximity to the potential donor kidneys.

Nilo Cold Box is obtainable from most camping equipment shops. Evazote—expanded Vinyl Acetate—from Expanded Rubber and Plastics Ltd., Mitcham Road, Croydon, Surrey.

—We are, etc.,

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1 Calne, R., *British Medical Journal*, 1969, **2**, 565.

Bleeding from Dialysis Shunt Sites

SIR,—I have frequently encountered the problem of persistent oozing of blood from the sites of newly created shunts in dialysis patients. These patients have usually been taking anticoagulant drugs, and oozing has persisted even when the wound has been explored for bleeding points and carefully resutured. Because of unwillingness to reverse the anticoagulation in these patients for fear of the shunt clotting, the following manoeuvre in order to arrest the haemorrhage has been developed.

The wound edges are infiltrated with 1-2 ml of bovine thrombin (200 NIH units/ml) and then a compression dressing applied for one hour. This simple technique has been used in three patients with complete success. Apart from some stinging at the site of injection, no complications have been encountered so far.—I am, etc.,

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Accidental Infection of Man with Mycoplasma caviae

SIR,—*M. caviae* is a species of mycoplasma only recently characterized[1] and apparently normally only found in guinea-pigs.[1,2] Attempts to transmit the organism under experimental conditions to a variety of laboratory rodents and lagomorphs have been unsuccessful, and even in guinea-pigs the organism failed to show any pathogenic properties. Details of these investigations will be published elsewhere.

During the course of this work, a very